Madecassoside

Cat. No.: HY-N0568

CAS No.: 34540-22-2

Molecular Formula: $C_{48}H_{78}O_{20}$

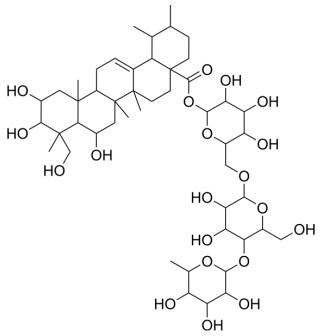
Molecular Weight: 975.12

Target: Endogenous Metabolite; Apoptosis; Autophagy; Keap1-Nrf2; p38 MAPK; Caspase

Pathway: Metabolic Enzyme/Protease; Apoptosis; Autophagy; NF-κB; MAPK/ERK Pathway

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (102.55 mM; Need ultrasonic)

H_2O : 33.33 mg/mL (34.18 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.0255 mL	5.1276 mL	10.2551 mL
5 mM	0.2051 mL	1.0255 mL	2.0510 mL
10 mM	0.1026 mL	0.5128 mL	1.0255 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 25 mg/mL (25.64 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (2.56 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (2.56 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (2.56 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Madecassoside is a pentacyclic triterpene isolated from Centella asiatica and has anti-inflammatory properties. Antioxidant and anti-aging effects.Madecassoside is a pentacyclic triterpene isolated from Centella asiatica. Madecassoside is orally active and has inhibitory properties against inflammation, oxidation, apoptosis and autophagy. Madecassosid inhibits activities of p38 MAPK and NF-kB[5][6], exhibits an anti-apopototic property, activates Nrf2 expression to reduce the

neurotoxicity[10]. Madecassoside can be used in endocrine diseases, cardiovascular diseases, skin diseases and other diseases.

In Vitro

Madecassosid (30, 100 μmol/L, 12 h) increases cell viability of oxidative injuried human umbilical vein endothelial cells (HUVECs)[5].
Madecassosid (10-100 μmol/L, 12 h) inhibits phosphorylation of p38 MAPK and activaty of Caspase-3, and therefore exhibits an antiapoptotic activity[5].
Madecassosid (10-100 μg/L) exhibits anxioxidative activity against melanocyte dendtrites retraction, maintaining mitochondrial membrane potential and Ca^{2+} homeostasis[7].
Madecassosid (30 μM) improves Insulin secretion by increasing expressions of p-IRS1, Akt and p-Akt under glucotoxic conditions[8].
Madecassosid (10 μM, 24 h) prevents inflammation and autophagy of neuronal cells induced by $A\beta_{25-35}$ through the class III PI3K/Beclin-1/Bcl-2 pathway[9].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[5]

Cell Line:	HUVECs
Concentration:	10, 30, 100 μmol/L
Incubation Time:	12 h
Result:	Increased cell viability to 68.9% and 78.3%, with concentration of 30 and 100 μmol/L, respectively.

Western Blot Analysis[5]

Cell Line:	HUVECs
Concentration:	10, 30, 100 μmol/L
Incubation Time:	12 h
Result:	Inhibited phosphorylation in p38 MAPK.

In Vivo

Madecassosid (6, 12, 24 mg/kg, i.v.) resolves neurological deficit and ameliorates neuronal apoptosis after focal cerebral ischemia reperfusion[6].
Madecassosid (6, 12, 24 mg/kg, i.v) inhibits activity of NF-kB and so prevents the brain injury[6].
Madecassosid (120 mg/kg, i.g.) reduces LPS-induced neurotoxicity and enhances heme oxygenase proteins via upregulation of Nrf2 in LPS-stimulated neurotoxicity[10].
Madecassosid (10-40 mg/kg,p.o.) ameliorates oxidative damage and imflammation after bleomycin (BLM) instillation, inhibits TGF-β1 overexpression and collagen synthesis, improves collagen degradation[11].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Focal cerebral ischemia reperfusion injury in Sprague Dawley rats[6]
Dosage:	6, 12, 24 mg/kg
Administration:	Intravenous injection
Result:	Reduced neurological deficit, infarct area, brain damage and neuronal apoptosis.

Animal Model:	Lipopolysaccharide-induced neurotoxicity in Sprague-Dawley rats[10]

Dosage:	30-120 mg/kg for 14 days
Administration:	Intraperitoneal injection
Result:	Reduced LPS-induced cognitive impairment and imflammatory cytokine, promoted Nrf2 pathway at concentration of 120 mg/kg.

REFERENCES

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